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The monoamine oxidase A gene as a potential moderator of the relationship between parental rearing and symptoms of borderline personality disorder in female undergraduates

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THE MONOAMINE OXIDASE A GENE AS A POTENTIAL MODERATOR OF THE
RELATIONSHIP BETWEEN PARENTAL REARING AND SYMPTOMS OF BORDERLINE
PERSONALITY DISORDER IN FEMALE UNDERGRADUATES

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ABSTRACT

THE MONOAMINE OXIDASE A GENE AS A POTENTIAL MODERATOR OF THE
RELATIONSHIP BETWEEN PARENTAL REARING AND SYMPTOMS OF BORDERLINE
PERSONALITY DISORDER IN FEMALE UNDERGRADUATES

Borderline Personality Disorder is characterized by intense emotional lability, resistance to treatment, interpersonal problems, and high rates of suicide. All of these result in extensive costs to individuals diagnosed with BPD, their loved ones, and to society in general. Yet there is still no general consensus concerning the relative importance of factors contributing to development of BPD. Linehan's 1993 biosocial model of BPD provides a framework for investigating factors such as biological vulnerabilities and invalidating environments. Although extreme versions of invalidation, such as childhood abuse, have received much attention, others like parental rearing styles have received limited attention. This is surprising as aversive parenting practices such as intrusive, erratic, punitive, and withholding emotions would seem pertinent determinants of an invalidating environment. Even more surprising is the paucity of research investigating potential interactions of parental rearing with genetic vulnerabilities, such as the Monoamine Oxidase A gene (MAOA). The present study pursued this line of investigation. Three variants of the MAOA gene were focused on. The highly active variant MAOA-H has been found in significantly higher rates among patients with Borderline Personality Disorder. The Monoamine Oxidase A gene creates a potential vulnerability, which in turn creates a potential pathway for Borderline

Personality Disorder development. Thus, the proposed study aimed to measure parental rearing as three constructs: a) overprotection, b) rejection, and c) emotional warmth. It was predicted that MAOA-H would moderate the effects of parental rearing on Borderline Personality Disorder symptoms by increasing the magnitude of the relationship between the three parental rearing styles and Borderline Personality Disorder symptoms, while MAOA-L would decrease it and MAOA-M would be intermediate. Three moderated hierarchical regressions were run to test these predictions. There was no moderation detected in any analyses. However, the predicted pattern of MAOA moderated relationships was confirmed for parental rejection and BPD symptoms, with the relationships being significant in the MAOA-H and MAOA-M groups. The relationship between overprotection and BPD symptoms was significant for the MAOA-H group, while in the MAOA-M and MAOA-L groups the relationships were both not significant. The relationship between emotional warmth and BPD symptoms was only significant for the MAOA-M group. While no moderation effect was revealed, the study was underpowered and a future replication study with a much larger sample may be able to detect similar trends while possessing the statistical power to detect moderation.

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The Monoamine Oxidase A Gene as a Potential Moderator of the Relationship Between Parental Rearing and Symptoms of Borderline Personality Disorder in Female Undergraduates

Borderline Personality Disorder (BPD) is associated with high rates of suicide, resistance to improvement with treatment, and high utilization of services resulting in costs to society (Leichsenring, Leibing, Kruse, New & Leweke, 2011). Prevalence rates of BPD have been reported at a range from 0.5% up to 6% of the general population. These prevalence rates are alarming when paired with the statistic that 10% of individuals diagnosed with BPD will commit suicide (Paris & Zweig-Frank, 2001). This rate is 50 times higher than the general population (Leichsenring et al., 2011).

BPD is broadly defined by emotion dysregulation, impulsivity, interpersonal difficulties, suicidality, non-suicidal self-injury, and identity disturbances. Understanding BPD and its etiology is very important and potentially the foundation for creating more favorable outcomes for individuals seeking treatment for BPD, yet there is still a lack of conclusiveness regarding the contributors to BPD symptoms. It is likely that BPD is not a disorder associated with a singular causal factor, but one characterized by many risk factors interacting with vulnerabilities to create and maintain the pathway to BPD symptoms. For example, BPD is heterogenous in that an individual diagnosed with BPD may only share one overlapping symptom with another individual diagnosed with BPD (Crowell, Beauchaine, & Linehan, 2009). The current study aimed to investigate the association between a specific biological vulnerability, namely, the MAOA gene, and the parental rearing variables of rejection, overprotection, and emotional warmth.

Linehan's 1993 biosocial model provides a strong framework for research on BPD. The biosocial model stresses an invalidating early environment as a contributor to the development of BPD within emotionally and biologically vulnerable individuals (Linehan, 1993). Invalidation can be conceptualized as excessive criticism, erratic responses, overinvolvement as well as a lack of recognition of the child's emotional state (Crowell et al., 2009). Children who are emotionally vulnerable have a susceptibility towards emotional dysregulation in the context of an invalidating environment. This emotional dysregulation is a key factor in the development and maintenance of BPD (Crowell et al., 2009). Vulnerable children who grow up in invalidating environments often resort to extreme emotional responses in order to obtain their needs, leading to the maintenance of the aversive environment and future emotional dysregulation (Linehan, 1993). This perpetuated emotional dysregulation has been found to serve as a mediating factor for BPD development as emotional dysregulation has been linked to key BPD behaviors such as aggression, self-harm, and suicide (Byrd et al., 2018). Thus, it is very important to focus research efforts on delineating the risk for BPD conveyed through various invalidating environments and biological vulnerabilities.

Much research has been directed towards the effects of extreme invalidation such as childhood abuse. Individuals diagnosed with BPD retrospectively report childhood abuse significantly more than healthy controls (Bandelow et al., 2005). Some have found childhood abuse to be reported from 30 to 60% percent of individuals diagnosed with BPD (Zanarini, 2000). The severity of childhood abuse is associated with BPD in such a manner that as the severity of abuse increases so does the severity of BPD symptoms (Zanarini et al., 2002), further solidifying an invalidating environment as a contributor to BPD symptoms. There is contention pertaining to specifically which types of childhood abuse convey the most risk for the

development of BPD (Hernandez, Arntz, Gaviria, Labad & Gutiérrez-Zotes, 2012), but the overarching conclusion based on the literature is that childhood abuse in general plays a significant role (Hernandez et al., 2012).

Parental Rearing

BPD is not a homogenous disorder and investigating contributors to BPD symptoms must continue to be sought. It is important not to focus on a singular event or factor as being causal, but to expand the investigation to other potential contributors to invalidating early environments. Parental rearing is a potential contributor that has received less attention than others such as childhood abuse. However, this lack of attention may not be warranted as a study by Nickel, Waudby, and Trull (2002) found aversive parental rearing to account for more of the relationship with BPD than childhood abuse.

Some studies classify parental rearing behaviors into three types: overprotection, emotional warmth, and rejection. Overprotection consists of, but is not limited to, intrusiveness, strict regulation, stringent monitoring, and protection against perceived negative experiences (Deković, et al., 2006). Parental rejection is conceptualized as hostility towards the adolescent through harsh or punitive punishment, criticism, and belittlement (Deković, et al., 2006). Emotional warmth is the degree to which the parents support their child and makes them feel loved (Arrindell et al., 1999). At face value these constructs as standalone factors may not appear aversive enough to contribute to the development of BPD, but an emotionally withdrawn, rejecting, yet intrusively overprotective parental rearing style may result in the invalidating environment described by Linehan's biosocial model (Crowell et al., 2009). Yet studies investigating the role of parental rearing variables have produced mixed results. It is not apparent

as to what pattern or possible interaction of these three rearing styles are associated with BPD symptoms.

In a study measuring perceived parental rearing styles in adolescents, only the variable of parental overprotection was associated with BPD symptoms (Schuppert, Albers, Minderaa, Emmelkamp & Nauta, 2012). Other studies report that low emotional warmth and high overprotectiveness are associated with BPD symptoms (Machizawa-Summers, 2007; Zweig-Frank & Paris, 1991). A study by Schuppert, Albers, Minderaa, Emmelkamp & Nauta (2015) found that less emotional warmth, high overprotection, and high rejection were all significantly associated with BPD symptoms. Nickel et al. (2002) found that parental rearing was more associated with BPD symptoms than adverse childhood experiences, such as loss or abuse. Despite these findings, there is disagreement regarding whether parental rearing is associated with BPD symptoms. For example, Hernandez et al. (2012) found that parental rearing had no significant association with BPD symptoms, whereas childhood abuse did.

Mixed outcomes may be due to several factors. Participants in these studies varied from one study to the next, ranging from inpatients, to outpatient patients, to university students. Although all studies purported to measure the same construct, the measures used to collect parental rearing data were not consistent. Self-report measures varied, which means the questions participants answered about parental rearing variables such as rejection, overprotection, and emotional warmth were inconsistent across studies, making it difficult to compare one study's results on parental rearing to another. In addition, the validity of retrospective reports may be subject to recall bias – that is, some investigators believe that individuals with BPD symptoms may be more likely to recall their past as more aversive in general, calling into question the validity of their retrospective reporting (Schuppert et al., 2015). Nevertheless, like the results of

retrospective studies, concurrent reports on parental rearing and prospective studies have found that aversive parental rearing practices are associated with BPD symptoms (Johnson, Cohen, Chen, Kasen & Brook, 2006; Nickel et al., 2002). Thus, it is important to continue researching BPD to establish both the internal and external validity of parental rearing to provide a more conclusive body of literature regarding early invalidating experiences and specifically which parental rearing styles convey risk for BPD symptoms.

Genetic Vulnerabilities

When Linehan originally formulated her biosocial model of BPD there was limited research on the biological vulnerabilities she referred to (Crowell et al., 2009). Since then the capabilities of researchers has increased substantially regarding genetics. For example, twin studies have provided foundational knowledge regarding heritability. Specifically, one twin study found an approximately 60% heritability for personality disorders in general (Torgersen et al., 2000). Additive genetic factors without any shared environments produced a 69% heritability rate for BPD (Torgersen et al., 2000). This indicates a strong genetic contribution for BPD. Going beyond looking at genetic contributions to heritability rates of BPD, Bornovalova et al. (2013) looked at the relationship between childhood abuse and BPD in a twin study design. The results illustrated that shared genetic factors accounted for a significant amount of the relationship between childhood abuse and BPD. Meta-analyses for twin and familial studies resulted in approximately a 40% heritability of BPD (Amad, et al, 2014), indicating that genetics plays a significant role in creating the biological vulnerability for BPD as described in Linehan's biosocial model. With the knowledge that genetic factors play a significant role in BPD, the next step is to look for candidate genes that convey the most vulnerability for BPD.

Further research has attempted to elucidate specific genes that influence BPD symptomology. These genes have included, but are not limited to, genes that regulate serotonin and dopamine. The serotonergic system when dysregulated is associated with emotional lability, impulsivity, and suicidality (Amad et al., 2014). A variety of genes act upon this system to alter its functionality. The dopaminergic system is similar in that it plays a role in emotion information processing, impulse control, and cognition (Crowell et al., 2009). Gene association studies have investigated genes that influence regulation of the serotonergic and dopaminergic systems separately. The results of these studies have been inconclusive in that no clear-cut candidate gene for BPD has been identified across these studies. A meta-analysis of specific gene relationships with BPD yielded no significant association (Amad et al., 2014). Perhaps the lack of clarity reflects low power due to relatively small sample sizes. Although there were some significant findings for individual studies, the meta-analysis failed to indicate a significant association across studies. The mixed results regarding candidate genes indicates that more work is needed.

Monoamine Oxidase A

The Monoamine Oxidase A gene (MAOA) regulates both the serotonergic system and dopaminergic system through the production of an enzyme that modulates levels of neurotransmitters (Crowell et al., 2009). There are three variants of this gene determined by the number of tandem repeats of a 30-base pair in the promoter region of the gene. A variation of the gene that produces high levels of the enzyme is designated as MAOA-H and another variant that produces low levels of the enzyme is designated as MAOA-L. Since the MAOA gene is located on the X-chromosome, a female can inherit both MAOA-H and MAOA-L genes. A process called X-inactivation randomly deletes one of these variants on a cell by cell basis, creating a

mixture of MAOA-H and MAOA-L in the same individual. Such cases are designated MAOA-M for present purposes. These distinctions among variants is important because the MAOA-H gene variant has been reported in BPD patients at significantly higher rates than healthy controls by Ni et al. (2007). More recently, it has been reported that there is an association between MAOA variants and personality pathology in women (Byrd et al., 2018). Specifically, these investigators found that MAOA-H was associated with higher levels of emotional dysregulation in those who experienced maltreatment as children and who were subsequently diagnosed with BPD. The present research seeks to determine whether there is evidence of a similar role for MAOA as a vulnerability factor moderating the relationship between parental rearing variables and BPD symptoms.

The Present Study

The primary aim of this study was to investigate the possibility that MAOA moderates the relationship between parental rearing variables of overprotection, emotional warmth, and rejection for BPD symptoms. My approach was to genotype MAOA in emerging adult females who provided retrospective reports of their parent's parental rearing behaviors and self-reported BPD symptoms. Information provided by environmental interaction with genetic biomarkers such as MAOA has the potential to inform treatment of BPD.

My principal hypothesis was that the MAOA gene would moderate the relationship between parental rearing and reports of BPD symptoms. Several specific predictions based on this hypothesis are:

Hypothesis 1: Parental overprotection and rejection would be positively associated with BPD symptoms.

Hypothesis 2: Parental emotional warmth would be negatively associated with BPD symptoms.

Hypothesis 3: The MAOA gene would moderate these relationships, evidenced by stronger positive associations of overprotection and rejection with BPD symptoms for MAOA-H than for MAOA-L. For MAOA-M, these positive associations with BPD symptoms would be intermediate between that of MAOA-H and MAOA-L.

Hypothesis 4: Emotional warmth would have a larger negative association with BPD symptoms in the presence of MAOA-H than in the presence of MAOA-L, with that of MAOA-M being intermediate.

Method

Participants

Data from a preexisting database was used for this study. The database included 148 female undergraduate volunteers from psychology courses at Eastern Washington University. Extra credit toward the student's final grade in their course was provided as an incentive for participation. Participants must have volunteered a useable DNA sample and completed all self-assessment items to be included in the analysis. Participant's ages ranged from 18 to 35 years old.

Design

The design was moderated linear regression with a polychotomous moderator variable. Parental rearing consisted of overprotection, rejection, and emotional warmth. Each moderated regression analysis employed one of the three parental rearing variables as an independent variable, BPD symptoms as a dependent variable, and the MAOA gene variants as the 3-valued moderator variable (MAOA-H, MAOA-L and MAOA-M).

Materials

s-EMBU

A shortened version of the Swedish EMBU was used to assess parental rearing styles (Arrindell et al., 1999; EMBU is a Swedish acronym for ‘my memories of upbringing’). The original scale had 81 items, but I used the shorter 23-item version (s-EMBU). There were three subscales for parenting style: overprotection (7 items), rejection (6 items) and emotional warmth (10 items). This scale uses a 4-point Likert scale (1 = no, never; 2 = yes, but rarely, 3 = yes, often 4 = yes, always). Participants could score anywhere from a 23 to a 92 on the s-EMBU

Overprotection consisted of 7 items pertaining to the participants parent’s intrusiveness, overinvolvement, anxiousness or fear for the participant’s safety. Parental rejection consisted of 6 items pertaining to shaming, punishment, and rejection via criticism of the participant. Emotional warmth was assessed using 10 items assessing perceived parental affection, encouragement, stimulation, and praise (Arrindell et al., 1999). The psychometric properties of the s-EMBU have been shown to be consistent with the widely used original form (Arrindell et al., 1999).

Borderline Symptom List

BPD symptoms were evaluated using the short version of the Borderline Symptom List (BSL-23). The original scale consisted of 95 items which was potentially too long to be practical in some research and clinical settings. The BSL-23 is made up of 23 of original 95 items. Thus, the BSL-23 serves as a convenient version of the original scale with comparable psychometric properties, sensitivity, and ability to distinguish BPD from other conditions (Bohus et al., 2009). Items on the BSL-23 consist of common complaints and impairments generally reported by BPD patients and are assessed on a 5-point Likert scale (0= not at all, 1 = a little, 2 = rather, 3 = much,

4= very strong). The items ask participants to assess how much they suffered from each symptom over the last week to obtain a present quantitative representation of BPD symptomology (Bohus et al., 2009). Participant scores can range from 0 to 93 on the BSL-23

Procedure

Participants were recruited for the larger original database of psychological and genetic variables through the online SONA system at Eastern Washington University. Each participant signed up for a timeslot online to come into the lab and complete the study. Upon arrival, participants were greeted by a research assistant and asked to sit at one of the three lab computers. SONA systems assigned participants a unique identification code that labeled their data in order to preserve anonymity. Participants completed the first half of a battery of surveys, including the s-EMBU. They then provided a DNA sample via a buccal swab. The participants self-administered the buccal swab by brushing the inside of each cheek ten times before placing the swab back in the test tube. Research assistants labeled participants DNA samples with corresponding identification codes and stored them in a refrigerator in the lab. The participants then returned to the computer to complete the remainder of the survey battery, including the BSL-23. DNA data was stored behind a locked door before transferring it to The Institute of Science and Technology in Spokane for genotyping. Survey data was stored on the password protected SONA system. The mean time to complete the study was 29 minutes.

DNA extraction and genotyping

DNA extraction was performed using the QIAamp DNA Buccal Cell Kit. The standard protocol recommended by the manufacturer of the kit was followed for extraction. DNA was quantified using readings at A260/A280/A320. Preliminary simple polymerase chain reaction

(PCR) assay was performed to assure that the DNA was amplifiable. DNA that was not amplifiable was discarded and corresponding data was not used in analysis.

MAOA-uVNTR sequences, used to identify MAOA variants, were identified using PCR and gel electrophoresis. These sequences were located between the Xp 11.23 and Xp 11.4 bands. Allele variations ranged from 2 repeats to 4 repeats. The 2 and 3 repeats both indicate the MAOA-L variant and were combined to represent a singular MAOA-L category. Variants with 3.5, and 4 repeats are indicative of the MAOA-H variant and were combined to represent a single MAOA-H category. Individuals who possessed both MAOA-H and MAOA-L, for example, a 3 repeat and a 4 repeat, were categorized as MAOA-M for the present study.

Results

Descriptive Statistics

Mean BPD scores are reported in Table 1 for each MAOA group. The mean scores for the three groups were very similar. Table 1 also reports the number of participants with BPD scores at least 1 standard deviation above the overall group mean. Given the variability in group sizes, a chi square test was performed to determine if the disparate frequencies for high-scoring participants were significantly different among the three MAOA groups, but they were not.

Table 1. Descriptive Statistics for BPD Symptoms

Statistics	All Groups	MAOA -L	MAOA-M	MAOA-H
Means	23.42	24.35	23.57	22.85
SD's	18.1	15.51	19.65	16.83
N	138	20	76	52
N > 1 SD	22	3	12	7
% > 1 SD	15.9%	15%	16%	13%

Correlations

Table 2 provides the results of Pearson correlations performed between each of the variables in this study without respect to genotype. The correlations between borderline personality symptom scores and both rejection and overprotection were significantly positive, as were the correlations between them. The correlations between borderline scores and emotional warmth were significantly negative, as was the correlation between emotional warmth and rejection. It is noteworthy that overprotection was not correlated with emotional warmth.

Table 2. Overall Correlation Matrix: BPD Symptoms and Parental Rearing

Variables	Statistics	BPD Symptoms	Rejection	Overprotection	Emotional Warmth
BPD Symptoms	Pearson r	1			
	N	148			
Rejection	Pearson r	.439**	1		
	N	138	138		
Overprotection	Pearson r	.291**	.465**	1	
	N	137	135	137	
Emotional Warmth	Pearson r	-.346**	-.499**	-0.073	1
	N	139	135	135	139

** Correlation is significant at the 0.01 level (2 tailed).

Tables 3, 4 and 5 report the Pearson correlations for the associations between parental rearing variables and each of the three genotypes. Table 3 indicates a lack of statistical significance between BPD symptoms and all parental rearing variables. Note, however, that the lack of statistical significance for these correlations may simply reflect low power attributable to relatively low frequencies in the MAOA-L group. Rejection and overprotection, however, are strongly and positively correlated despite the low N. The correlation between emotional warmth and rejection is strong and negative.

Table 3. MAOA-L Correlation Matrix: BPD Symptoms and Parental Rearing

Variables	Statistics	BPD Symptoms	Rejection	Overprotection	Emotional Warmth
BPD Symptoms	Pearson r	1			
	N	20			
Rejection	Pearson r	0.213	1		
	N	20	20		
Overprotection	Pearson r	0.332	.603**	1	
	N	19	19	19	
Emotional Warmth	Pearson r	-0.171	-.638**	-0.157	1
	N	19	19	18	19

** Correlation is significant at the 0.01 level (2-tailed).

Table 4 reports the correlations between parental rearing variables and BPD symptoms for the MAOA-M group. The correlation between BPD symptoms and rejection was significant and positive while the correlation between BPD symptoms and emotional warmth was significant and negative. Furthermore, the correlation between rejection and overprotection was positive and significant with the relationship between rejection and overprotection being negative and significant.

Table 5 reports the correlations between parental rearing variables and BPD symptoms for the MAOA-H group. BPD symptoms were significantly and positively correlated with both rejection and overprotection. Rejection was positively and significantly correlated with overprotection. Emotional warmth and rejection were negatively and significantly correlated.

Table 4. MAOA-M Correlation Matrix: BPD Symptoms and Parental Rearing

Variables	Statistics	BPD Symptoms	Rejection	Overprotection	Emotional Warmth
BPD Symptoms	Pearson r	1			
	N	76			
Rejection	Pearson r	.372**	1		
	N	70	70		
Overprotection	Pearson r	0.188	.431**	1	
	N	69	68	69	
Emotional Warmth	Pearson r	-.475**	-.538**	-0.078	1
	N	72	69	69	72

** Correlation is significant at the .01 level (2 tailed)

Table 5. MAOA-H Correlation Matrix: BPD Symptoms and Parental Rearing

Variables	Statistics	BPD Symptoms	Rejection	Overprotection	Emotional Warmth
BPD Symptoms	Pearson r	1			
	N	52			
Rejection	Pearson r	.620**	1		
	N	48	48		
Overprotection	Pearson r	.455**	.506**	1	
	N	49	48	49	
Emotional Warmth	Pearson r	-0.212	-.431**	-0.071	1
	N	48	47	48	48

** Correlation is significant at the 0.01 level (2-tailed).

Fisher's r to z transformation was used to assess if BPD symptoms correlation with the parental rearing variables would differ significantly between MAOA groups. The analysis revealed that there was a significant difference between the MAOA-H and MAOA-L group correlations for parental rejection and BPD symptoms $z = 1.75$, $p = .04$. No other correlations were significantly different from each other.

Moderated Regressions

Three hierarchical regression analyses were performed using the Process macro in SPSS (Hayes & Rockwood, 2020). A different parental rearing variable was used for each analysis – specifically, rejection, overprotection, or emotional warmth. Parental rearing served as the independent variable, genotype as a polychotomous moderator variable, and BPD symptoms as the dependent variable.

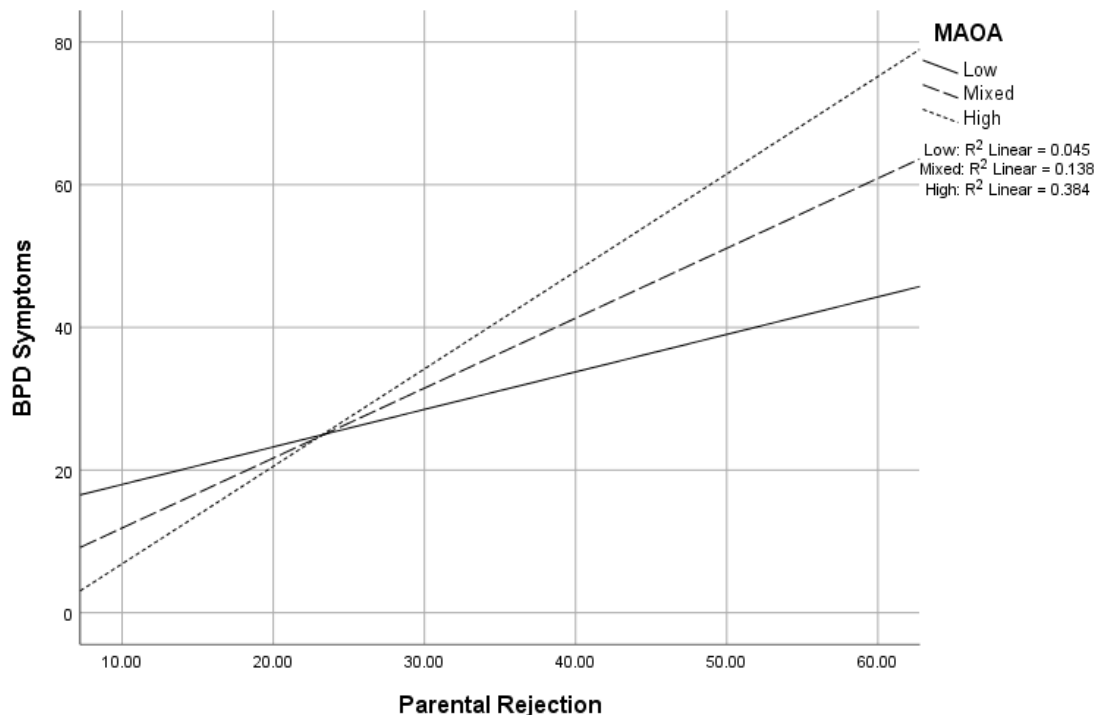
Rejection. To test the hypothesis that the relationship between parental rejection and BPD symptoms is moderated by the MAOA gene, a two-step hierarchical multiple regression analysis was performed. In the first step, rejection and MAOA were the independent variables and borderline scores were the dependent variable. This regression did not include an interaction term and will be referred to hereafter as Model 1. Model 1 accounted for a significant proportion of variance in BPD symptoms, $R^2 = .193$, $F(2, 135) = 16.14$, $p < .001$. Model 1 revealed a main effect of parental rejection, $\beta = .439$, $t(135) = 5.67$, $p < .001$ with rejection predicting higher BPD symptoms, but there was no main effect for MAOA. Model 2 added an additional term to the regression analysis to determine whether a potential interaction between parental rejection and MAOA would improve prediction.

Model 2 also accounted for a significant portion of variance in BPD symptoms, $R^2 = .204$, $F(5, 131) = 6.77$, $p < .001$, but the R^2 change was not significant $\Delta R^2 = .011$, $F(2, 132) = .93$, $p = .40$, indicating a lack of a MAOA moderation effect. Moreover, in contrast to Model 1, the main effect of parental rejection was not significant in this model, apparently because some participants with missing data were excluded listwise in this analysis. This reflects a difference in the SPSS protocol for handling missing cases in more complicated analyses, such as Model 2

(missing data is handled pairwise in simpler analyses such as correlation and simple linear regression).

Figure 1 illustrates the overall direction of the relationship between parental rejection and BPD symptoms. In addition, Figure 1 also depicts the simple slopes relating parental rejection to BPD symptoms. Although Model 2 found neither a significant main effect for parental rejection nor a significant interaction involving genotype, two of the simple slopes, representing the conditional effect of rejection on BPD, depicted in Fig 1 were significantly different from zero and both were positive, $\beta = 0.98$, $t(132) = 3.68$, $p < .001$ for MAOA-M; and $\beta = 1.37$, $t(132) = 4.04$, $p < .001$ for MAOA-H. The simple slope for MAOA-L, $\beta = .88$, though apparently positive, was not significantly different from zero.

Figure 1. Simple Slopes for BPD Symptoms and Parental Rejection



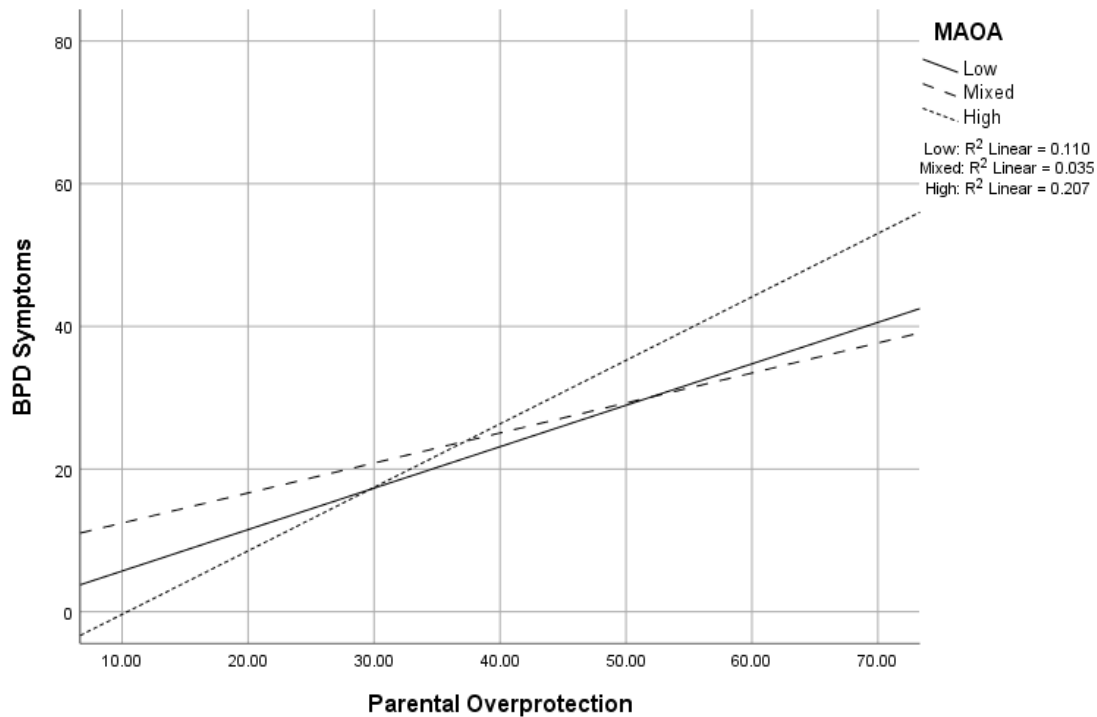
Overprotection. A two-step hierarchical multiple regression analysis was also conducted to test the hypothesis that the MAOA gene would moderate the relationship between parental

overprotection and BPD symptoms. The first step included parental overprotection and BPD symptoms as independent variables which accounted for a significant proportion of variance in BPD symptoms $R^2 = .09$, $F(2,134) = 6.28$, $p = .002$. There was a main effect of parental overprotection $\beta = .601$, $t(134) = 3.54$, $p = .001$ with higher overprotection predicting higher levels of BPD symptoms. However, there was no main effect of MAOA on BPD symptoms.

For model 2 an interaction term between overprotection and MAOA was added to assess if the interaction term would significantly increase the predictability of the model. This model also accounted for a significant proportion of variance in BPD symptoms $R^2 = .098$, $F(5,131) = 2.84$, $p = .02$. However, the addition of the interaction term did not account for significantly more variance than without the interaction term $\Delta R^2 = .01$, $F(2,131) = .93$, $p = .40$. The main effect for parental overprotection was not significant in the second model. Again, this is likely due to the loss of cases that occurred between model 1 and model 2 described in the earlier moderated regression analysis.

Figure 2 illustrates the simple slopes for the moderated regression analysis between overprotection and BPD symptoms. The direction of the relationship between overprotection and BPD symptoms for each MAOA group is shown. Although the simple slopes for MAOA-L and MAOA-M were indistinguishable and not significantly different from zero, that for MAOA-H was significantly positive, $\beta = 0.89$, $t(131) = 3.10$, $p = .002$.

Figure 2. Simple Slopes for BPD Symptoms and Parental Overprotection



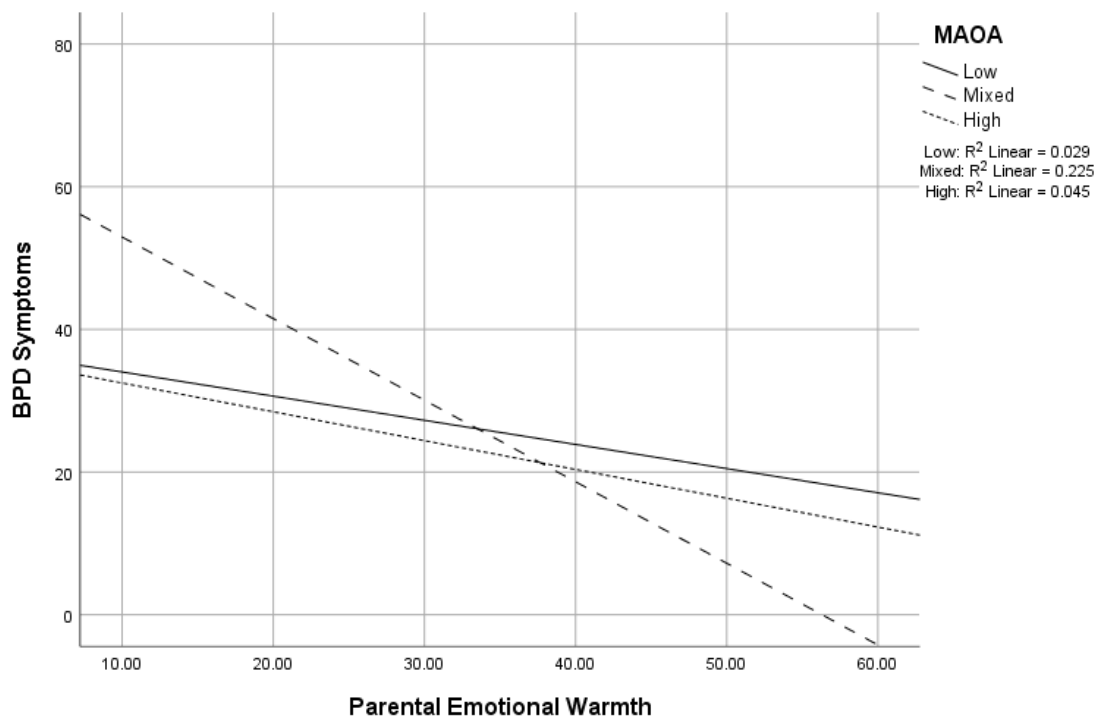
Emotional Warmth. Finally, A two-step hierarchical multiple regression analysis was performed to assess if MAOA moderates the relationship between emotional warmth and BPD symptoms. In the first step, emotional warmth and MAOA were included as independent variables. Emotional warmth and MAOA accounted for a significant proportion of variance in BPD symptoms $R^2 = .13$, $F(2,136) = 9.74$, $p < .001$. There was a main effect of emotional warmth on BPD symptoms with lower emotional warmth predicting higher BPD symptoms $\beta = -.77$, $t(136) = -4.39$, $p = < .001$. There was no main effect of MAOA on BPD symptoms.

The interaction term for emotional warmth and BPD symptoms was then added into the model to determine if the interaction term would significantly increase the predictability of the model. While the second model did account for a significant amount of variance in BPD symptoms $R^2 = .16$, $F(5,133) = 4.89$, $p = .004$, the addition of the interaction term did not significantly increase the variance accounted for in BPD $\Delta R^2 = .03$, $F(2,133) = 2.36$, $p = .10$. The

main effect of emotional warmth was not significant in the second model $\beta = -.34$ $t(133) = -.64$, $p = .53$. Similar to the other analyses, there was a drop in cases between model 1 and model 2.

Figure 3 illustrates the simple slopes for the moderated regression between emotional warmth and BPD symptoms. The conditional effect of emotional warmth on BPD symptoms was significant for the MAOA-M group $\beta = -1.14$, $t(133) = -4.65$, $p < .001$. There were no significant conditional effects of emotional warmth on BPD symptoms for MAOA-H or MAOA-L.

Figure 3. Simple Slopes for BPD Symptoms and Parental Emotional Warmth



Discussion

My principal hypothesis was that the MAOA gene would moderate the relationship between parental rearing and reports of BPD symptoms. A second objective was to investigate the relationship between the parental rearing variables and BPD symptoms.

The significant positive correlations between parental rejection and symptoms of BPD as well as that between overprotection and symptoms of BPD are consistent with Hypothesis 1. In

addition, the significant negative correlation between emotional warmth and symptoms of BPD are consistent with Hypothesis 2. These findings support the general hypothesis that BPD symptoms are associated with, and perhaps determined in part by, parental rearing styles. Although it appears that the association between the rearing variable of rejection is stronger than that of overprotection, the difference in correlations was not significant, $Z_H = 1.81$, $p = 0.07$. It is noteworthy, however, that parental overprotection was not correlated with emotional warmth.

This further affirms that aversive parenting is associated with BPD symptoms and possibly the etiology of BPD. Therefore, when researchers attempt to operationalize the invalidating early environment described by Linehan (1993) it would be beneficial to take into consideration parental rearing behaviors. It also warrants further investigation into parental rearing as a primary contributor to BPD. While severe forms of maltreatment may be the primary contributor to BPD my results suggest that parenting that is characterized by harsh punishment, low acceptance, stringent monitoring, and low support creates a potential environment that contributes to increased risk for BPD symptomology. Further investigation may help to elucidate exactly what parenting behaviors contribute to an invalidating environment and which of these behaviors confer the greatest risk for BPD.

Moderator Analysis: MAOA and Parental Rejection

The simple slopes depicting the association of rejection and BPD symptoms for the three genotypes (Figure 1) are consistent with Hypothesis 3 – i.e. the simple slopes were in the predicted order. Furthermore, the positive slopes for MAOA-M and MAOA-H were significantly positive. The slope for MAOA-L, though also positive, was not significantly different from zero. While these outcomes for Model 2 are suggestive, they do not confirm Hypothesis 3 because the apparent interaction did not account for a statistically significant improvement in the variance

accounted for in Model 2 relative to Model 1, thus it is not possible to conclude that MAOA moderates the relationship between parental rejection and BPD symptoms. Given the relatively low N in the MAOA-L group, this outcome might reflect a lack of statistical power.

Moderator Analysis: MAOA and Parental Overprotection

Despite the overall positive association between overprotection (Figure 2), the simple slopes relating overprotection to BPD symptoms were not in the predicted order for the three genotypes. Although the slope for MAOA-H was significantly positive, the slopes for MAOA-L and MAOA-M were essentially indistinguishable and not significantly different from zero. Moreover, as was the case with rejection, Model 2 did not yield a significant improvement in the proportion of variance accounted for relative to Model 1, consequently there was no MAOA moderator effect, and so Hypothesis 3 was not confirmed.

Moderator Analysis: MAOA and Parental Emotional Warmth.

The relationship between parental emotional warmth and BPD symptoms was expected to be negative, or, to put it another way, the lower the emotional warmth, the greater expected BPD symptoms. Such a relationship is in fact apparent (Figure 3), but the order of simple slopes for the genotypes was not expected. Hypothesis 4 predicted an intermediate slope for MAOA-M, but the simple slope for this genotype was not only the steepest of the three genotypes, but it was the only slope significantly different from zero. Taking this outcome at face value, it is as though the mixed genotype confers risk for BPD symptoms when emotional warmth is not available from the parent and is protective for BPD symptoms when emotional warmth is available from the parent. This outcome was not predicted and, additionally, as was the case with both rejection and overprotection, Model 2 did not bring a significant improvement in the proportion of

variance accounted for. Despite the interesting outcome with MAOA-M, there was no moderator effect and consequently Hypothesis 4 was not confirmed.

Summary and Conclusions

Preliminary correlation analyses revealed a trend that appeared to tentatively support the prediction that MAOA-H would confer the greatest risk for BPD symptoms. The correlations between the parental rearing variables and BPD symptoms were larger in the MAOA-H group than the other groups, except for the correlation between emotional warmth and BPD symptoms in the MAOA-M group. However, the only significantly different correlations were between parental rejection and BPD symptoms, with the MAOA-H group's correlation being larger than the MAOA-L group's correlation. This was in line with prior studies that identified MAOA-H as the risk variant for BPD symptoms (Byrd et al., 2018).

The simple slopes for the emotional warmth moderated regression analysis were surprising as MAOA-M appeared to pose the greatest risk for increasing BPD symptoms in the presence of low emotional warmth. This could be due to the MAOA-M group containing a mixture of both MAOA-H and MAOA-L. However, when looking at the simple slopes for the moderated regression analyses between overprotection and BPD symptoms and rejection and BPD symptoms it appears that a pattern emerged with the MAOA-H variant conferring the greatest risk. The overall trend of the slopes would suggest that as rejection and overprotection increase those with the MAOA-H variant are at the most risk to develop greater BPD symptoms, making MAOA-H the risk variant except for in the case of emotional warmth. This is similar to the result that Byrd, et al (2018) found showing that women with the MAOA-H variant who experienced early childhood maltreatment had greater increases in emotion dysregulation and subsequent greater levels of BPD symptom severity compared to the other variants.

Furthermore, the simple slopes of the parental rearing variables and BPD symptoms for each MAOA variant were meaningful, suggesting that both MAOA-H and MAOA-M act as a biological vulnerability for increased BPD symptomology in the presence of aversive parenting. When looking at the slopes, MAOA-H appears to have the steepest slope and therefore potentially confers the most risk for BPD symptoms when in a rejecting or overprotective environment. However, MAOA-M confers the greatest risk when there is a lack of emotional warmth. Thus, it might be that in most cases MAOA-M does not have as strong of an effect as MAOA-H, but the fact that an individual possesses a mixture of MAOA-H and MAOA-L still puts them at greater risk than only possessing the MAOA-L variant.

With a larger sample size potentially leading to a significant moderation effect, the trends of my results would coincide with the greater literature on MAOA and BPD. MAOA has been shown to have an interactive effect with other serotonergic genes to increase risk for BPD (Ni et al., 2009). Other studies have found MAOA-H to occur at significantly higher rates in BPD patients compared to healthy controls (Ni et al., 2007). It has also been shown that MAOA-H interacts with environmental factors, such as childhood maltreatment, to contribute to BPD etiology (Byrd et al., 2018). Contrary to the trends of the present study, a study by Yang et al. (2014) found a significantly higher frequency of MAOA-L in patients dependent on opioids with BPD compared to patients dependent on opioids without BPD. Although this study found a different variant to confer risk for BPD symptoms, it still implicates MAOA as a contributor to BPD etiology. This literature in combination with the trends that the simple slopes and correlations revealed provides a strong case for there to be a potential moderator effect of MAOA on the relationship between parental rearing and BPD symptoms in a study that has a large sample size.

It is also possible that MAOA does not moderate the relationship between parental rearing and BPD symptoms. To my knowledge this is one of the first studies to look specifically at MAOA as a moderator of the relationship between parental rearing variables and BPD symptoms. Most gene by environment studies have looked at how salient contributors to BPD such as childhood abuse interact with MAOA to contribute to BPD etiology. It might be the case that parental rearing is not as salient of a contributor as childhood abuse and therefore does not interact with MAOA in a similar way to modulate BPD symptomology. Essentially, parental rearing might not have a large enough effect on BPD symptoms for the gene by environment interaction to be significant.

Many of the studies I cited used populations, such as inpatient BPD patients, that have much higher BPD symptomology than the sample in the present study. The participants in the present study were from a college population and most of them would likely score much lower on the BSL-23 than an individual diagnosed by a clinician for BPD. In fact, only 15.9% of individuals in the total sample scored 1 standard deviation above the mean for BPD symptoms. This could also contribute to the inability to detect a moderation effect as the individuals who reported high levels of BPD symptoms in the present study's sample were scarce.

However, to elucidate whether the trends this study identified are indicative of moderation, future studies should increase the sample size significantly and look to increase the number of participants that are recruited with high symptomology. Gene by environment studies require a large sample size as the variance of the interaction term, which represents the moderation effect, is often much smaller than the main effects of the gene or environmental factors. An analysis of 103 gene by environment studies showed that the median sample size for these studies was 345, which is regarded as underpowered to detect the variance that the

interaction terms account for (Duncan & Keller, 2011). The sample size in the current study was less than half of what was considered underpowered by Duncan & Keller (2011). Thus, if the direction and pattern of the results remained the same, with a much larger sample a replication study may be able to detect significant moderation.

The current study only looked at the MAOA gene as a moderator but there are many other genes that could have been taken into consideration. The serotonergic system has been identified as playing a potentially important role in BPD (Crowell et al., 2009). Genes that regulate the serotonin system such as 5-HT2C and TPH2 have been shown to be associated with BPD (Ni et al., 2009). In order to truly elucidate a gene by parental rearing interaction it would have been conducive to include these into the study. Including a broader range of genes would further facilitate the identification of genetic vulnerabilities. For example, Ni et al. (2009) included a broad range of genes into their study and were able to identify that MAOA interacted with three other serotonergic genes to influence BPD etiology (Ni et al., 2009). Without taking into consideration a broader range of genes the present study may not have been capturing the total picture on biological vulnerabilities. To truly reveal the molecular mechanisms of a complex disorder like BPD it is important to take into consideration multiple gene-gene and gene-environment interactions to elucidate potential impacts on the etiology of BPD. If future studies take these into consideration, then the complex disorder of BPD can be further understood, and the details of how biological vulnerabilities interact with one's environment to contribute to the development of BPD can be further delineated.

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